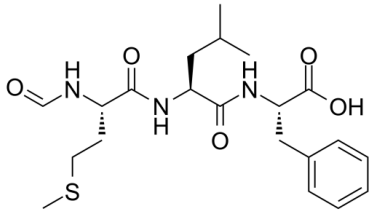
Data Sheet

Product Name:	N-Formyl-Met-Leu-Phe
Cat. No.:	CS-6489
CAS No.:	59880-97-6
Molecular Formula:	C21H31N3O5S
Molecular Weight:	437.55
Target:	TNF Receptor
Pathway:	Apoptosis
Solubility:	DMSO : ≥ 82.5 mg/mL (188.55 mM)



BIOLOGICAL ACTIVITY:

N-Formyl-Met-Leu-Phe (fMLP; N-Formyl-MLF) is a chemotactic peptide and a specific ligand of N-formyl peptide receptor (FPR). N-Formyl-Met-Leu-Ph is reported to inhibit **TNF-alpha** secretion. IC50 & Target: TNF-alpha[1] **In Vitro:** Binding of N-Formyl-Met-Leu-Phe to its specific cell surface receptor, N-formyl peptide receptor (FPR), triggers different cascades of biochemical events, eventually leading to cellular activation. FPR is a chemoattractant receptor belonging to the G protein-coupled receptor family. N-Formyl-Met-Leu-Phe promotes osteoblastic commitment and suppresses adipogenic commitment under osteoblastic differentiation conditions. N-Formyl-Met-Leu-Phe stimulates osteogenesis is associated with increased expression of osteogenic markers and mineralization. N-Formyl-Met-Leu-Phe inhibits expression of peroxisome proliferator-activated receptor-γ1. N-Formyl-Met-Leu-Phe-stimulated osteogenic differentiation is mediated via FPR1-phospholipase C/phospholipase D-Ca^{2+}-calmodulin-dependent kinase II-ERK-CREB signaling pathways[1]. N-Formyl-Met-Leu-Phe, a bacterial-derived peptide, induced proinflammatory cytokine gene expression in human peripheral blood monocytes. Bacterial products LPS and N-Formyl-Met-Leu-Phe synergistically induce inflammatory response via multiple signaling pathways. TLR4, IKKβ-IκBα, and NF-κB signaling pathways are involved in the synergistic induction of TNF-α via p65 nuclear translocation-dependent mechanisms[2]. **In Vivo:** N-Formyl-Met-Leu-Phe promotes bone formation in zebrafish and rabbits. Extensive skeletal development is evident at 5 dpf in over 80% of N-Formyl-Met-Leu-Phe-treated zebrafish. Treatment with N-Formyl-Met-Leu-Phe results in increased expression of Runx2. Bone marrow spaces are widely formed, and connective tissue covering bone is dense, like periosteum, in N-Formyl-Met-Leu-Phe-treated calvaria[1]. N-Formyl-Met-Leu-Phe mediate release of calprotectin from PMN in vitro. It induces release of calprotectin from PMN in a dose dependent manner. A minimum of 10% of total PMN calprotectin is retained at concentrations of 0.1-10.0 nM of N-Formyl-Met-Leu-Phe[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]Cells are cotransfected with either a dominant negative form of IκBα or a dominant negative form of IKKβ together with the NF-κB-dependent luciferase reporter plasmid. The plasmid pCMVβ is used as a control for transfection efficiency and this is monitored via the expression of β-galactosidase. Cells are transiently transfected with plasmids using DEAE-dextran. The transfected cells are cultivated for 48 h before a 6-h incubation in medium ±N-Formyl-Met-Leu-Phe, LPS, or N-Formyl-Met-Leu-Phe/LPS. Luciferase activity is determined by using the luciferase assay kit and a Monolight 3010 luminometer[2]. **Animal Administration:** [2] Mice: N-Formyl-Met-Leu-Phe is prepared in sterile PBS. Under the anesthesia, mice are intranasally treated with LPS (0.3 mg/kg) or N-Formyl-Met-Leu-Phe (0.5 mg/kg) or N-Formyl-Met-Leu-Phe and LPS in 50 μL of sterile PBS (control), BAL is performed by cannulating the trachea with sterilized PBS, and cells from BAL fluid are stained with Wright-Giemsa stain after cytocentrifuge. For TNF-α protein release, BAL fluid is collected and secreted TNF-α is measured by ELISA as described above[2].

References:

[1]. Shin MK, et al. N-formyl-methionyl-leucyl-phenylalanine (fMLP) promotes osteoblast differentiation via the N-formyl peptide receptor 1-mediated signaling pathway in human mesenchymal stem cells from bone marrow. J Biol Chem. 2011 May 13;286(19):17133-43.

[2]. Chen LY, et al. Synergistic induction of inflammation by bacterial products lipopolysaccharide and fMLP: an important microbial pathogenic mechanism. J Immunol. 2009 Feb 15;182(4):2518-24.

[3]. Hetland G, et al. Chemotaxins C5a and fMLP induce release of calprotectin (leucocyte L1 protein) from polymorphonuclear cells in vitro. Mol Pathol. 1998 Jun;51(3):143-8.

CAIndexNames:

L-Phenylalanine, N-formyl-L-methionyl-L-leucyl-

SMILES:

O=C(O)[C@H](CC1=CC=CC=C1)NC([C@H](CC(C)C)NC([C@H](CCSC)NC=O)=O)=O

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